"ASSESSMENT OF MITRAL VALVE RESISTANCE INDEX BY ECHOCARDIOGRAPHY IN MITRAL STENOSIS BEFORE AND AFTER BALLOON MITRAL VALVOTOMY AND ITS HEMODYNAMIC IMPLICATIONS"

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In partial fulfillment of the requirements
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D.M. BRANCH - II CARDIOLOGY

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CERTIFICATE

This is to certify that the dissertation entitled **"ASSESSMENT OF MITRAL VALVE RESISTANCE INDEX BY ECHOCARDIOGRAPHY IN MITRAL STENOSIS BEFORE AND AFTER BALLOON MITRAL VALVOTOMY AND ITS HEMODYNAMIC IMPLICATIONS"** is the bonafide original work of **Dr.M.RAJENDRAN,** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2013.The period of postgraduate study and training was from August 2010 to July 2013.

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DECLARATION

I, **Dr.M.RAJENDRAN**, solemnly declare that this dissertation entitled, **"ASSESSMENT OF MITRAL VALVE RESISTANCE INDEX BY ECHOCARDIOGRAPHY IN MITRAL STENOSIS BEFORE AND AFTER BALLOON MITRAL VALVOTOMY AND ITS HEMODYNAMIC IMPLICATIONS"** is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2010 – 2013 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor V.E.Dhandapani M.D.D.M. This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology**.

Dr.M.RAJENDRAN

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CONTENTS

INTRODUCTION

Valvular stenosis is common cardiac disease with greater morbidity and mortality especially in developing countries like India. Echocardiography is considered as an important and simple tool to evaluate valve stenosis .Almost all cases of mitral stenosis are due to rheumatic heart disease.

Assessment of severity of mitral stenosis by echocardiography utilizes many parameters using 2D echo, M mode, and Doppler methods. Conventional methods include mitral valve orifice area determination by planimetry and pressure half time method, pressure gradient determinations with Bernoulli's equation, and mitral leaflet separation index. But the common problem that occurs in all these measurements is that, only anatomic information alone is provided to the clinician.

In most of the situations, clinical decisions are made by assessing the functional or hemodynamic status of the valve lesions irrespective of the choice of management. This fact is further strengthened by many observations that, for given mitral valve orifice area, different hemodynamic profiles found to be present.

Hence therapeutic judgments have to be contemplated based on functional or hemodynamic status of the patients.

Mitral valve resistance index is newer tool that describes mitral stenosis in physiological terms. This newer index is calculated by the formula as given below

$$MVR = TMMG/Q \times 1333.$$

MVR is mitral valve resistance, Q is trans mitral blood flow rate, and 1333 is used to convert resistance into dynes.cm$^{-5.}$. Q is calculated by the expression of stroke volume in terms of diastolic filling period. Since mitral valve resistance index incorporates both duration of trans mitral flow and quantity of blood flow across the valve, it negates the disadvantage faced by the other parameters like pressure gradients. The hemodynamic burden of mitral stenosis is reflected by the pulmonary artery pressure as the symptoms of stenotic lesions closely parallel the magnitude of pulmonary arterial hypertension.

On the other hand, the estimation of pulmonary artery pressure using Doppler estimation of systolic pressure gradient between the right atrium and right ventricle, explains the hemodynamic burden but not the severity of stenosis. The degree of

pulmonary arterial hypertension is quantified by adding right atrial pressure to the right ventricular systolic pressure. In case of aortic stenosis, hemodynamic burden of the lesion was well correlated with aortic valve resistance as suggested by some studies.

In our study , we sought to assess the valve resistance index in mitral stenosis before and after balloon commissurotomy and to observe the correlation between PASP and other parameters with valve resistance.

AIM OF THE STUDY

1) To assess mitral valve resistance index in mitral stenosis by echocardiography before and after balloon mitral valvotomy.

2) To study the relationship between systolic pulmonary artery pressure and mitral valve resistance both before and after percutaneous mitral commissurotomy.

3) To study the correlation between severity of mitral stenosis and mitral valve resistance.

REVIEW OF LITERATURE

ANATOMY

The mitral apparatus is composed of five components. [annulus, leaflets, commissures, chordae tendineae, and papillary muscles) (Fig-1). In order to perform normal function, all the parts of valve need to act in coordinated fashion in addition to optimal atrio-ventricular function .Suboptimal function of the valve apparatus results from any abnormality of these parts in isolation or combination. The mitral valve annulus forms a complete fibrous ring that is strongly attached to the circumference of the anterior mitral leaflet by the strong fibrous skeleton of the heart [1] . The peculiarity of mitral valve in respect to other heart valves is the presence of only two leaflets. Being hemispherical and larger, the anterior leaflet, also forms the part of outflow tract of left ventricle. The posterior mitral leaflet is rectangular and is usually divided into three scallops. The middle scallop is the largest of the three in more than 90 percent of normal hearts. The anterior leaflet is twice the height of the posterior leaflet but has half its annular length.[1] With advanced age, the mitral leaflets thicken somewhat, particularly along their closing edges.[2] The commissures are cleft-like splits in the leaflet tissue that represent the sites of

separation of the leaflets . left ventricle gives rise to origin of anterolateral papillary muscle and posteromedial papillary muscle beneath the commissures. Chordate tendinae connect the papillary muscle to the free edge of

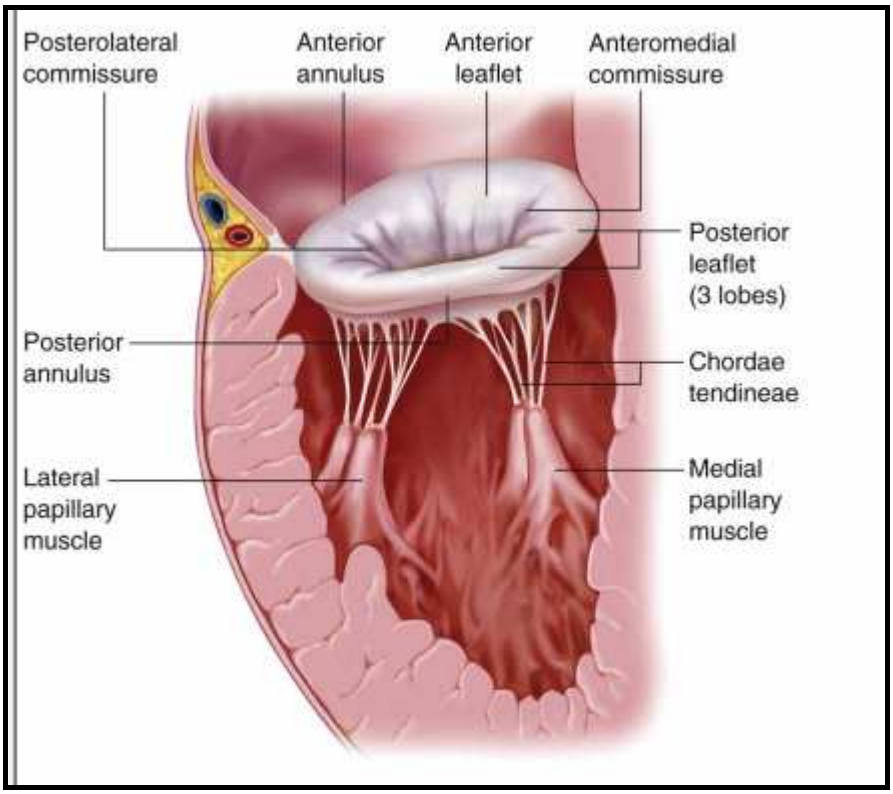


Figure-1; Shows the Normal Anatomy of Miral Valve

The leaflets close to the commissures. (major commissures)[1]. The anterolateral papillary muscle is usually solitary and has a dual blood supply from the left coronary circulation. In contrast, the posteromedial papillary muscle usually has multiple heads and is most commonly supplied only by the right coronary artery.[3]. Contraction of papillary muscle aids in closure

of valve leaflets. The line of closure for either mitral leaflet is not its free edge but an ill-defined junction between a thin, clear zone and a thicker, rough zone[1] Unlike the tricuspid valve, the normal mitral leaflets have no chordal insertions into the ventricular septum.[3] The functional orifice of the mitral valve is defined by its narrowest diastolic cross-sectional area. This can be at the annulus when there is extensive annular calcification or close to the papillary muscle tips in populations with rheumatic mitral valve obstruction.

MITRAL STENOSIS

Mitral stenosis is mechanical impedance to ventricular filling as a result of narrowing of mitral valve orifice, which results in restricted ventricular filling. The most common cause for mitral stenosis is rheumatic heart disease, accounting for 99% of causes. Past history of rheumatic fever is usually absent in more than 50% of patients, and, at least half of the populations with rheumatic fever will not develop rheumatic heart disease. About 25% of all patients with rheumatic heart disease have isolated MS, and about 40% have combined MS and MR. Multivalve involvement is seen in 38% of MS patients, with the aortic valve affected in about 35% and the tricuspid valve in about 6%. The pulmonic valve is rarely

affected. The molecular mimicry between streptococcal antibodies and epitopes of valve tissues by possessing same M protein, holds the key for the pathological basis of rheumatic fever. Frequent inflammations of the valves with scarring and fibrosis results in mitral stenosis eventually .[4]Other causes of left ventricle inflow obstruction are extremely rare, which includes congenital mitral stenosis, left atrial myxoma, mitral annular calcification ,ball valve thrombus, and mucopolysacharidoses. Mitral annular calcification usually accompanies calcification of aortic valve as a part of atherosclerosis.[5].

Between 1940 and 1970 rheumatic heart disease accounted for the majority of heart diseases. After the widespread use of penicillin the incidence of rheumatic fever significantly declined in developed countries. But in developing countries like India still RHD is a major economic and health burden, due to overcrowding, poor hygiene and lack of access to better health care

Figure 2; Showing the typical fish mouth appearance of mitral valve in mitral stenosis

. In India, currently 6 million people are suffering from RHD. Mitral stenosis is the sine quo non of established rheumatic valvular pathology. It happens to be the predominant valvular heart disease of rheumatic heart disease. It causes greater morbidity and mortality. There is a substantial fall in the incidence of rheumatic valve disease in western societies, in part due to widespread use of penicillin and improved socio economic status. In woods serious the latency period from ARF until the onset of symptoms of MS was 19 years. Isolated mitral stenosis occurs in 40% of patients. In India, critical MS may be present in children as young as 6 to 12 years old. In North America and Western Europe, however,

symptoms develop more slowly and occur most commonly between the ages of 45 and 65 years. The most likely causes for these differences are the relative prevalence of rheumatic fever and lack of primary and secondary prevention in developing countries, resulting in recurrent episodes of valve scarring

The normal mitral valve area is 4to 6 cm^2.Rheumatic process in mitral stenosis leads to thickening, and reduced mobility of leaflets, fusion of commissures and chordae and calcification or combination of these features. Commissural fusion results in narrowing of primary mitral orifice, whereas chordal fusion narrows secondary orifice. The symmetrical fusion of the commissures results in a small central oval orifice in diastole that on pathologic specimens is shaped like a fish mouth (Fig-2) or buttonhole because the anterior leaflet is not in the physiological open position . With end-stage disease, the thickened leaflets may be so adherent and rigid that they cannot open or shut, reducing or, rarely, even abolishing the first heart sound and leading to combined MS and MR. When rheumatic process involves subvalvular damage primarily rather than inflammation of commissures, dominant mitral regurgitation will ensue more likely. In mitral stenosis, development of diastolic pressure gradient is

essential for forward propulsion of blood from left atrium to left ventricle.[6]. In response to progressive increase in pressure gradient, the left atrium enlarges and elevated left atrial pressure is transmitted to the pulmonary veins and capillaries.

HEMODYNAMICS

It is very clear that maintaining cardiac output with the small valve area requires a higher gradient and thus an elevated LA pressure. Similarly, an increased need for cardiac output, such as occurs during exercise or pregnancy, results in an increase in gradient and high LA pressures. Little is the effect of the length of the diastolic filling period on the relation between cardiac output and gradient. The time available for systole is that part of the cardiac cycle occupied by isovolumic contraction and relaxation or by ejection. As the heart rate increases, the total amount of time spent during systole increases despite a reduction in the systolic time per beat. Thus, time available for diastole decreases as the heart rate increases. Because blood can flow through the mitral valve only during diastole, the flow rate is inversely proportional to the duration of the flow period at a constant stroke volume. Of course, a higher flow rate results in a greater loss of energy to friction and requires a larger gradient and higher LA pressures

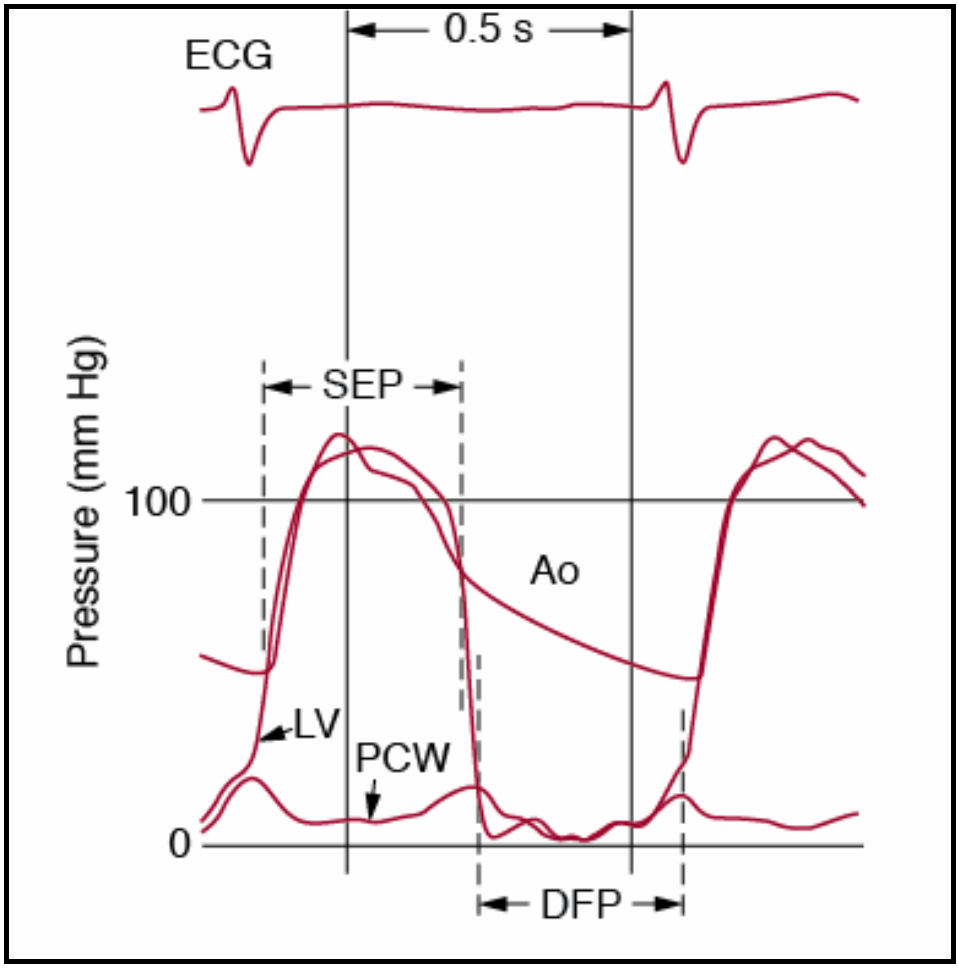


Figure -3 Shows the normal pressure tracings of left atrium (LA), left ventricle (LV), and aortic (AO) pressures. DFP=diastolic filling period; SEP=systolic ejection period.

It is important to remember that the gradient from LA to LV is a function per beat, not per minute. Thus, the gradient is dependent on the stroke volume and the diastolic filling time, as well as the LV diastolic pressure (Fig 3and 4). Stroke volume output from left ventricle is reduced as mitral valve stenosis escalates further. This clinical syndrome of lung congestion and declined cardiac output resembles left sided failure. In 30% of patients with mitral stenosis left ventricle ejection fraction is

reduced in response to decline in preload of left ventricle and secondary vasoconstriction due to fall in cardiac output, rather than poor contractile function. [7] In up to 20 percent of patients, the pulmonary vascular resistance is also elevated, which further increases PA pressure. PA hypertension results in hypertrophy and enlargement of right ventricular chamber . The changes in right sided ventricular function eventually result in right atrial hypertension and enlargement and systemic venous congestion; frequently, tricuspid regurgitation also occurs. Pulmonary venous hypertension alters lung function in several ways. Distribution of blood flow in the lung is altered, with a relative increase in flow to the upper lobes, and, therefore, in physiologic dead space. Pulmonary compliance generally decreases with increasing pulmonary capillary pressure, increasing the work of breathing, particularly during exercise. Chronic changes in the pulmonary capillaries and pulmonary arteries include fibrosis and thickening. These changes protect the lungs from the transudation of fluid into the alveoli (alveolar pulmonary edema).

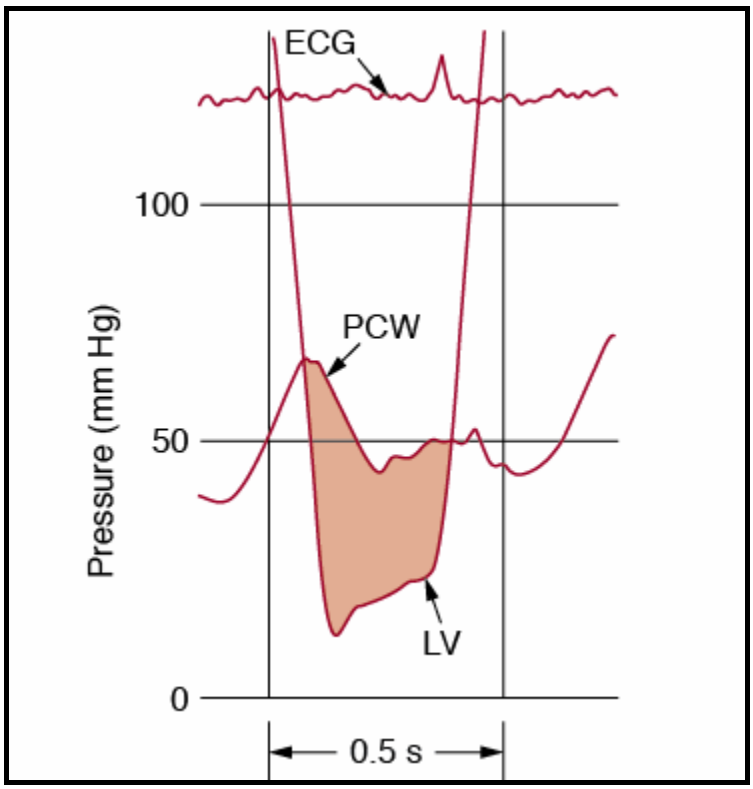


Figure 4 –Showing the diastolic pressure gradient between left ventricle and left arium in mitral stenosis.

Indeed, it is not uncommon to find patients with severe MS whose resting PA wedge pressure (indirect LA pressure) exceeds 25 to 30 mmHg. Capillary and alveolar thickening, which help protect against pulmonary edema, further add to the abnormalities of ventilation and perfusion. Pulmonary vascular changes cause an elevated pulmonary vascular resistance.

In some patients with high pulmonary vascular resistance and right ventricle (RV) dysfunction, cardiac output may be low. The body maintains oxygen consumption by extracting more oxygen from the arterial blood, and the mixed venous oxygen content falls.

The hemoglobin-O2 dissociation curve is shifted to the right, facilitating the unloading of oxygen from hemoglobin to the tissues. The reduced cardiac output may result in a surprisingly small gradient across the mitral valve despite severe stenosis. Although pulmonary congestion may be less striking in these patients, the cardiac output does not increase normally with exercise, and, typically, the patients are severely limited by fatigue.

Long-standing MS with severe PA hypertension and resultant RV dysfunction may be accompanied by chronic systemic venous hypertension. Tricuspid regurgitation is frequently present, even in the absence of intrinsic disease of this valve. Functional pulmonic regurgitation may also be present. Dependent edema formation and visceral congestion directly reflect elevated systemic venous pressure and salt and water retention. Chronic passive congestion in the liver leads to central lobular necrosis and eventually to cardiac cirrhosis

COMPLICATIONS

Late diastolic augmentation of pressure gradient between left atrium and left ventricle up to 30% occurs during atrial systole in mitral stenosis. AF is common in patients with MS, with an increasing prevalence with age. In patients with severe MS younger

than 30 years, only about 10% are in AF compared with approximately 50% of those older than 50 years. Inadequate atrial contraction during atrial fibrillation is known to reduce left ventricle stroke volume by 20%, and setting the stage for occurrence of symptoms.

Obstruction at the mitral valve level has other hemodynamic consequences, which account for many of the adverse clinical outcomes associated with this disease. Passive pulmonary arterial hypertension results from transmission of left atrial pressure through pulmonary veins with normal pulmonary vascular resistance. This in turn followed by reactive pulmonary arterial hypertension, characterized by elevated pulmonary vascular resistance and pulmonary artery pressure. This in turn has profound effect on right heart function.. In addition, left atrial enlargement and stasis of blood flow is primarily responsible for heightened threat of thrombus formation and systemic embolism. Typically, the left ventricle is relatively normal, unless there is coexisting MR, with the primary abnormalities of the left ventricle being a small underfilled chamber and paradoxical septal motion caused by RV enlargement and dysfunction.

HEMODYNAMIC PROGRESSION

Serial echocardiographic data have described the rate of hemodynamic progression in patients with mild MS.[8-10]. The overall rate of progression was a reduction in mitral orifice area of 0.09 cm2/yr. Approximately one third of patients showed rapid progression, defined as a fall in valve orifice area greater than 0.1 cm2/yr.

ASSESSMENT OF SEVERITY OF MITRAL STENOSIS BY ECHOCARDIOGRAPHY

Echocardiography is an important tool for diagnosing and evaluating vale stenosis. It is also a popular non invasive tool of choice for assessment of valvular stenosis. Therapeutic judgment depends on echo based evaluation of the severity of valve obstruction, so it is important to have universal standards in order to maintain accuracy and consistency when evaluating and providing the final report of valve stenosis. The severity of mitral stenosis can be evaluated using 2D echo and Doppler methods. Mitral valve orifice area is commonly used to measure the severity of obstruction without the influence of loading conditions. Many methods are available to calculate MVOA [mitral valve orifice area], but none is fully satisfactory. The following methods are used.

1) Planimetry of MVOA

2) Pressure Gradient

3) Pressure Half Time

4) Continuity Equation

5) Pisa-proximal Isovelocity Surface Area Method

PLANIMETRY

Planimetry of mitral valve orifice area utilizing 2D ECHO offers the merit of being a direct measurement of valve area and not influenced by loading conditions, atrio-ventricular compliance or associated valvular lesions, unlike other modalities. 2D imaging of the mitral orifice area using planimetry is best correlated with anatomical valve area as validated with catheterization and surgical derived values. . Hence planimetry is considered as the reference measurement for mitral valve area[11,12].Planimetry of valve area is done by direct tracing of inner border of mitral valve opening in mid diastole, including opened commissures using parasternal short axis view.

To ensure mitral valve area measurement at the tip of leaflets, meticulous scanning from apex to bas of left ventricle is needed.

The measurement should be done perpendicular to the mitral orifice. Attention should be paid on echo machine settings such as gain, transmission power as these may affect the image. Gain should be such that the whole contour should be visualized. Higher gain setting will overestimate and lower gain will under estimate valve area. Planimetry of the area using zoom mode is essential in order to better delineate the inner border of mitral orifice. Use of harmonic imaging for the purpose of improvement of planimetry measurement is not clear

LIMITATIONS OF PLANIMETRY

1. Significant leaflet tip calcification

2. Poor border definition

3. Highly deformed valve due to commissurotomy

4. Eccentric orifice

5. Highly operator dependent, not feasible in 5% of population even in experienced echo cardiographers.

In the presence of atrial fibrillation, and incomplete commissural fusion it is advocated to do many different measurements. 3D echo and 3D guided biplane imaging will

enhance optimal positioning of measurement plane even with less expertise in echocardiography.

PRESSURE GRADIENT

Doppler echo helps in assessment of diastolic pressure gradient between left atrium and left ventricle, as a non invasive tool. The transvalvular gradient can be calculated by continuous wave Doppler of mitral inflow during diastole with the help of velocity spectrum using modified Bernoulli equation ie $P= 4V^2$.This is considered reliable because , it correlates better with invasive measurement using transseptal catheterization.[13]. The use of continuous wave Doppler is better suited to ensure peak velocities are included. In case of pulse-wave Doppler, the sample volume be ideally located at the level of leaflet tips. Apical 4chamber view is better suited for assessing Doppler gradient because of parallel orientation, obtained with blood flow. Under estimation of velocities can be prevented by proper orientation of beam with the flow. It is wise to use colour Doppler in locating eccentric jets, usually a result of severe minor and major chordal disease. In these cases, the Doppler beam is guided by the highest flow velocity zone identified by colour Doppler. Attention should be paid on instrument settings by changing gain, acquiring good window and

proper orientation of beam in order to get ideal Doppler spectrum with clearly made out margins. Peak and mean gradients of the valve are estimated, with the assistance of software, using the continuous wave Doppler waveforms on the monitor. It is considered that mean gradient is ideally suited as a hemodynamic marker. (Figure 7). In view of influence of atrial and ventricular compliance on peak mitral velocity, from which peak gradient is derived, peak gradient is less desired parameter as a matter of fact.[14] Heart rate definitely deserves a mention while reporting gradients as it affects the gradient. In patients with atrial fibrillation, mean gradient estimation must be done by averaging of five cycles with the least variation of R–R intervals and similar to normal heart rate. Mean gradient has its important prognostic value after balloon mitral valvotomy.

The correlation between mean gradient and other echocardiographic parameters should always be checked before judging the magnitude of obstruction, especially planimetry of valve area and pressure half time methods.

DEMERITS

1) Even though mean trans mitral gradient correlates well with left atrial pressure obtained through transseptal

catheterization, it poorly correlates with mitral valve gradient measured using pulmonary capillary wedge pressure.

2) Mean gradient is also influenced by transvalvular flow rate. Overestimation of severity in high out put states and mitral regurgitation, and vice versa.

3) Underestimation of mean gradient also occurs if the angle of interrogation of beam with the mitral flow is greater than 30º. This is overcome by using colour flow Doppler with continuous Doppler.

4) Aortic regurgitation jet, a high velocity jet, may contaminate the mitral stenotic jet and mean gradient may tend to be over estimated, especially when eccentric. On the other hand ,trans mitral gradient may be underestimated when significant aortic regurgitation elevates left ventricular diastolic pressure.

PRESSURE HALF TIME

Pressure half time is defined as the time required for the peak pressure gradient to reach its half level and is the the same for peak velocity to decrease to a velocity equal to peak velocity divided by √2[=1.4]. There is a negative correlation between fall in transmitral

velocity and the mitral valve area (cm2), and MVA is derived using the empirical formula:[15]

$$MVA=220/T1/2$$

Pressure half time is estimated by tracing the deceleration slope of the E-wave on Doppler spectral display of transmitral flow valve. Software embedded in the echo machine calculates the valve area. A good Doppler signal is obtained with proper orientation of beam with blood flow. One should be mindful about the deceleration slop, which is sometimes bimodal, due to unequal fall in mitral blood flow velocity in relation to duration of diastolic period. It is advocated in such situation, to start tracing from mid diastole and tracing from early part of diastole is to be avoided. [16]**.** If one encounters a velocity spectrum with a concave shape, it is better not to use pressure half time method to assess mitral valve stenosis severity, even though very rarely encountered. On the other hand, pressure half time calculation in atrial fibrillation requires echocardiographer to skip the short cycle lengths in addition to averaging several cardiac cycles to obtain meaningful estimation.

It has been well established that the factors that affect the deceleration slope of E wave are transmitral diastolic pressure gradient during early diastole, compliance of left atrium and ventricular chamber filling properties, in addition to mitral valve area. These observations were originally made from applying principles of hydro dynamics to simulation models and in vitro transmitral flow models. [15,17]

The number 220 used in pressure half time calculation actually represents an empirical constant which in turn reflects the product of atrio-ventricular compliance and square root of peak gradient, without considering ventricular relaxation.. In most studies, pressure half time method has a good correlation with mitral area because of rise in mean gradient is usually counteracted by decrease in compliance. But this is not a case when the mean gradient and compliance undergo sudden changes. Pressure half time estimation with in 48 to 72 hrs after percutaneous transmitral balloon valvotomy is affected by disproportionate changes between net compliance and mean gradients.[18]

.A pressure half time of 220 msec means a valve area of 1cm2. A pressure half time of 440 means 0.5 cm2. The software present in the echo machine will calculate the pressure half time. In

case of bimodal deceleration slope, tracing should begin from mid diastole. On the other hand ,if the slope is concave, measurement is not possible.

MERITS

1) It is easy to perform

2) It is not influenced by heart rate, cardiac output.

3) Mitral regurgitation does not affect the accuracy of pressure half time.

DEMERITS

1) Pressure half time is affected by altered compliance of left atrium and left ventricle.

2) Pressure half time is not an ideal method to assess severity of mitral stenosis up to 72 hrs of balloon mitral valvotomy.

3) In concave shaped velocity spectrum, it is not feasible to measure pressure half time

CONTINUITY EQUATION

It is the gorlin formula of the echocardiography, which can be used as tool to calculate the area of stenotic lesion as well as

regurgitant lesions. It works on the concept of conservation of flow, literally means what comes in must go out.(Fig-4 (a))

Because the flow rate (or volume) is the product of the area and velocity (or TVI) of flow, a stenotic or regurgitant orifice area can be calculated from measurements of flow and flow velocity.

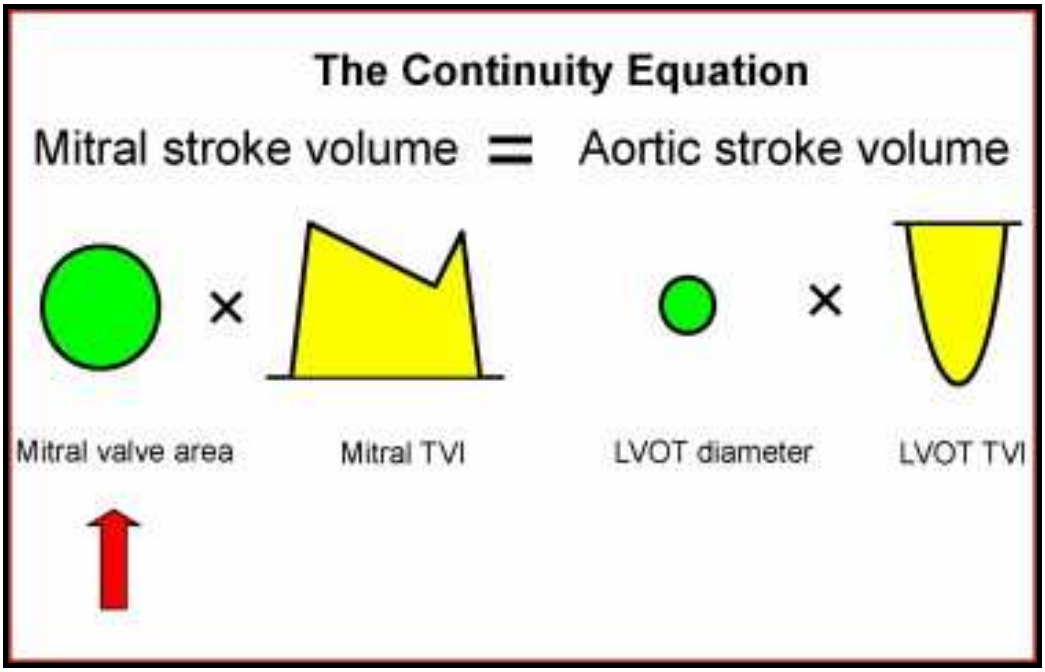


Figure-4 (a) Shows calculation of continuity equation; LVOT=left ventricular outflow tract; TVI= time velocity integral

Flow across a stenotic or regurgitant orifice is the same as a proximal (or upstream) flow across a known area and velocity. Hence,

$$A1 \times TVI_1 = A2 \times TVI^2$$

where A1 is a known area at a location proximal to the unknown area, A2. TVI is measured with pulsed wave or continuous wave Doppler echocardiography.

$$A_2 = A_1 \times \left(\frac{TVI_1}{TVI_2}\right)$$

In aortic stenosis, flow across the aortic valve area (A2) is the same as flow across the LVOT (A1). In mitral regurgitation, flow across the regurgitant mitral valve orifice (A2) is the same as flow at a PISA (A1). It should be noted that the ratio of the areas is inversely proportional to their TVI ratio:

$$MVA = \Pi[D^2/4][VTI_{AORTIC}/VTI_{MITRAL}]$$

DEMERITS

1) Poor reliability compared with other parameters

2) Less useful in atrial fibrillation and presence of significant mitral or aortic regurgitation.

PISA METHOD

Flow convergence or proximal isovelocity surface area method calculates transmitral flow rate. Convergence of blood flow occurs in a series of isovelocity hemispheres when it passes through a narrowed orifice.

MVA=Π(r2)(Valiasing)／Peak Vmitral ·ά／1800

Where r is the radius of the convergence hemisphere (in cm), Valiasing is the aliasing velocity (in cm/s), peak VMitral the peak CWD velocity of mitral inflow (in cm/s), and a is the opening angle of mitral leaflets relative to flow direction.[622]This method is useful in mitral regurgitation.

DEMERITS

1) Flow convergence is influenced by geometric complexities of mitral valve orifices**.**

2) Operator skill dependent and several estimations warranted.

3) Poor reliability of measurement of radius of flow convergence

4) Rarely used in mitral stenosis due to the use of single colour image denoting a fraction of diastolic duration, and the rest of diastole not studied.

NEWER ECHOCARDIOGRAPHIC TOOLS

1. Mitral Leaflet Separation Index

This index is calculated by measuring the distance between tips of two mitral leaflets in two orthogonal views. Some studies suggest, this has better correlation with planimetry of mitral valve.

It is much easier to measure with rapidity and can be a complementary tool.

2. Mitral Valve Resistance Index

This is a newer echocardiographic tool and defined as the ratio of mean mitral gradient to diastolic filling rate which in calculated by stroke volume divided by diastolic filling period. It is considered to be an alternative measurement of severity of mitral stenosis. Since it has better correlation with pulmonary artery pressure than other tools, it is considered to be better indicator of hemodynamic status of mitral valve obstruction.

BALLOON MITRAL VALVOTOMY

Percutaneous balloon mitral valvotomy predominantly uses inoue balloon. It works on the principle of splitting of commissures. On average there is 80% increase in mitral valve area after valvotomy. Usually more than half of the initial diastolic gradient is reduced after mitral balloon valvotomy. (Fig-5)

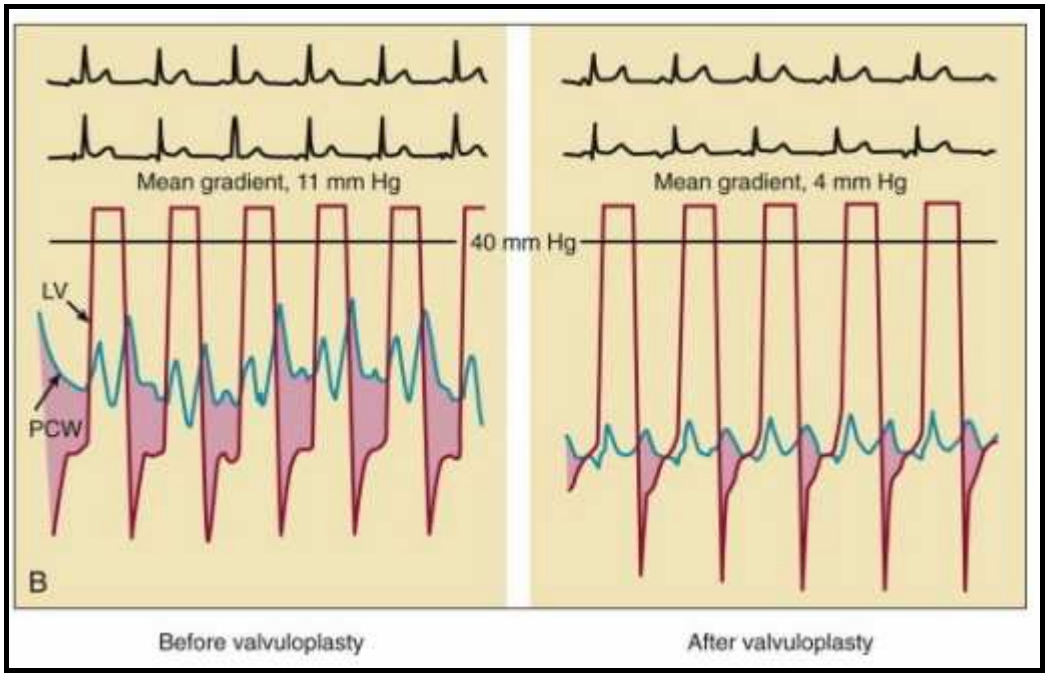


Figure-5 Shows the decline in diastolic gradient after balloon mitral valvotomy.

More than 50% increase in valve area noted in most studies. Pulmonary artery pressure decline takes longer time after valvotomy compared with other parameters. Balloon valvotomy is comparable to open mitral valvotomy and superior to closed mitral commissurotomy as suggested by studies.

METHODOLOGY OF THE STUDY

STUDY DESIGN

This study has been confirmed by the Ethic Committee of Madras medical college, Tamilnadu Dr MGR medical University and all the participants were informed of its objectives before the study and signed a letter of consent in accordance with the Helsinki Declaration Standards.

PATIENTS PROFILE

This is a prospective cohort study.During a period of 6 months, 20 patients with pure Mitral Stenosis who were referred and eligible for percutaneous commissurotomy of mitral valve who agreed to undergo 2D and Doppler echocardiographic examination. It was made sure that they have an adequate tricuspid regurgitant jet for systolic PAP calculation was detectable both before and after valvotomy, were prospectively recruited in this study.

INCLUSION CRITERIA

1) Patients with symptomatic rheumatic mitral stenosis undergoing balloon mitral valvotomy.

2) Adequate tricuspid regurgitation jet.

EXCLUSION CRITERIA

1) LA thrombus

2) More than mild MR, AS, AR and pulmonary stenosis

3) CAD requiring surgical revascularization

4) Poor echocardiographic window

5) Critically ill patients.

6) Organic tricuspid valvular disesase.

ECHOCARDIOGRAPHIC MEASUREMENT

Echocardiography was performed by only one operator using Philips HD 7 instrument in left lateral position, which has a 3.5 MHZ transducer and is capable of M-mode, 2D and Doppler study. Echocardiographic examinations carried out just before and 72 hrs after mitral balloon valvotomy. For all patients at least 2 or 3 measurements in sinus rhythm and at least 5 measurements in atrial fibrillations were taken in standard 2D and Doppler methods. In this study we have used the following methods;

1) 2D echo

2) M mode echo

3) Continuous wave doppler

4) Pulse wave doppler

5) Colour doppler

In order to avoid confounding factors influence, wall motion abnormalities were excluded in all patients by eye balling method, for calculating stroke volume. The stroke volume was calculated using product of cross sectional area of LVOT and time velocity integral of LVOT, in apical 4 chamber view. Left atrial diameter with maximum value was taken in parasternal long axis view in antero posterior dimension. Mitral valve orifice area by planimetry was estimated in parasternal short axis view, with scanning from ape to base to obtain entire contours and narrowest area, with adjusted gain settings in mid diastole.

Mitral valve orifice area by pressure half time method was calculated using continuous Doppler with optimum velocity spectral contour. In case of bimodal spectrum, we measured from mid diastole. We avoided the concave shaped slopes in mitral jets for the estimation of pressure half time. We have measured both mean and peak gradients of continuous Doppler velocity spectrum, but used mean gradient only as it is ideal in case of mitral stenosis.

We have also measured diastolic filling period with the help of pulse wave Doppler in seconds.

Pulmonary artery systolic pressure was estimated by using tricuspid regurgitation jet and RVSP[right ventricle systolic pressure] was calculated by Bernoulli's equation. Right atrial pressure was assumed to be 10mmhg in all patients.

MITRAL VALVE RESISTANCE

Mitral valve resistance is calculated by the following formula.

$$MVR = TMMG/Q \times 1333$$

MVR is mitral valve resistance; TMMG is trans mitral mean gradient, Q means trans mitral flow rate.1331 is used to convert resistance value in terms of dynes.cm^{-5}. Q is calculated by dividing the stroke volume by diastolic filling period ie

Q[trans mitral flow rate]= stroke volume / diastolic filling period.

All these echocardiographic variables were calculated before and after 72 hrs of mitral balloon commissurotomy.

RESULTS

DEMOGRAPHIC FEATURES

Of the 29 patients enrolled for the echocardiographic study, 5 patients excluded because of poor echo window and 6 patients not included for study due to poor tricuspid regurgitation jet..

Table-1 Descriptive status of baseline echo variables before PTMC

variables	Minimum	Maximum	Mean	Std. Deviation
AGE[years]	25	45	32.60	6.021
MVA(P)[mmhg]	.7000	1.4000	1.020000	.2015728
MVA[PHT]cm2	.7000	1.5000	1.065000	.2368099
MEAN GRADIENT[mmhg]	8.9	21.0	14.975	3.3529
LVOT VTI[cm]	17.7000	28.2000	20.940000	3.3443432
LVOT DIA[mm]	16	21	17.80	1.673
STROKE VOLUME[ml/s]	35.5	96.0	52.175	14.1340
DFP[msec]	282	556	440.00	62.790
PASP[mmhg]	39	96	58.75	15.099
VRI[dynes.cm-5	34.5	331.0	182.475	68.5138

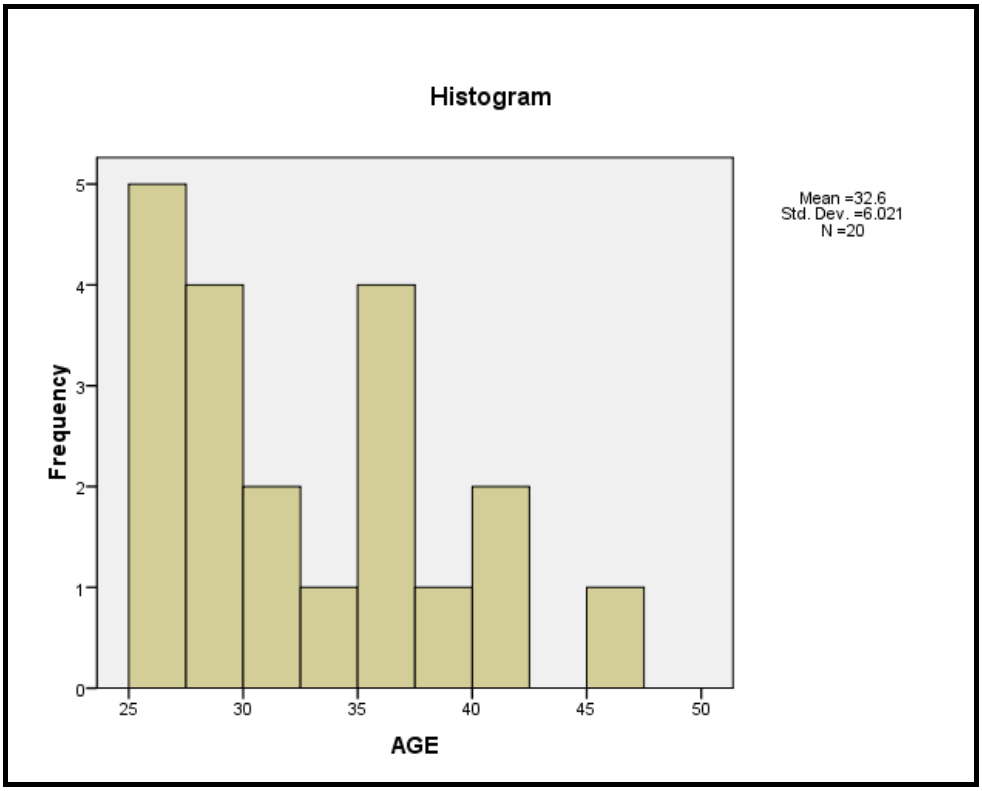
Finally 20 patients of mitral stenosis who were eligible for balloon valvotomy have undergone routine echocardiogram Examined patients were aged from 25 to 45 years with a mean age of 32.6±6 as observed in table 1. Also female patients outnumbered male patients, constituting 12 out of 20 patients, as well depicted by the pie chart[FIG-2].

Table-2; Descriptive status of echo characteristics after PTMC.

VARAIBLES	Minimum	Maximum	Mean	Std. Deviation
LEFT ATRIAL DIAMETER[cm]	2.5000	4.8000	3.975000	.5972525
MVA(P) cm2	1.2	2.0	1.590	.2315
MVA[PHT]	.6000	2.5000	1.650000	.4466248
MEAN GRADIENT[mmhg]	3.2	11.0	7.335	1.7113
LVOT VTI[cm]	18.7	29.0	23.485	3.3700
LVOT DIA[mm]	16.0	22.0	18.515	2.0035
STROKE VOLUME[ml/s]	41.0	111.0	64.240	15.9042
DFP[msec]	267	580	432.55	80.401
PASP[mmhg]	36	71	49.40	10.323
MVR[dynes.cm-5]	14.0000	172.0000	74.045000	35.3463685

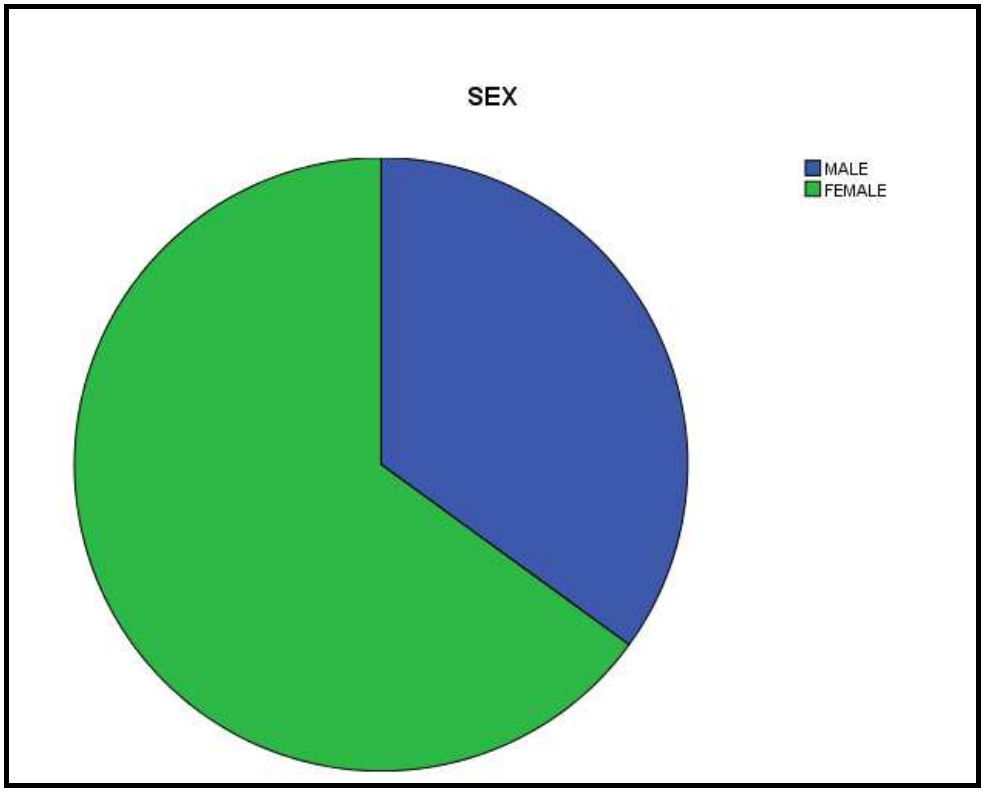
MVA=mitral valve area=planimetry, PHT=pressure half time, LVOT= left ventricle outflow tract,VTI=velocity time integral,DFP=diastolic filling period, PASP=pulmonary artery systolic pressure, and MVR=mitral valve resistance.

Figure-6; Age wise distribution of patients with mitral stenosis eligible for balloon mitral valvotomy.



Age of the patients ranged from 25 years to 45 years with mean age of 32.6 yrs, which reflect younger age of onset of complications secondary to mitral stenosis necessitating percutaneous valve interventional therapy.

Figure-7.: Pie chart representing female predominance among balloon mitral valvotomy.

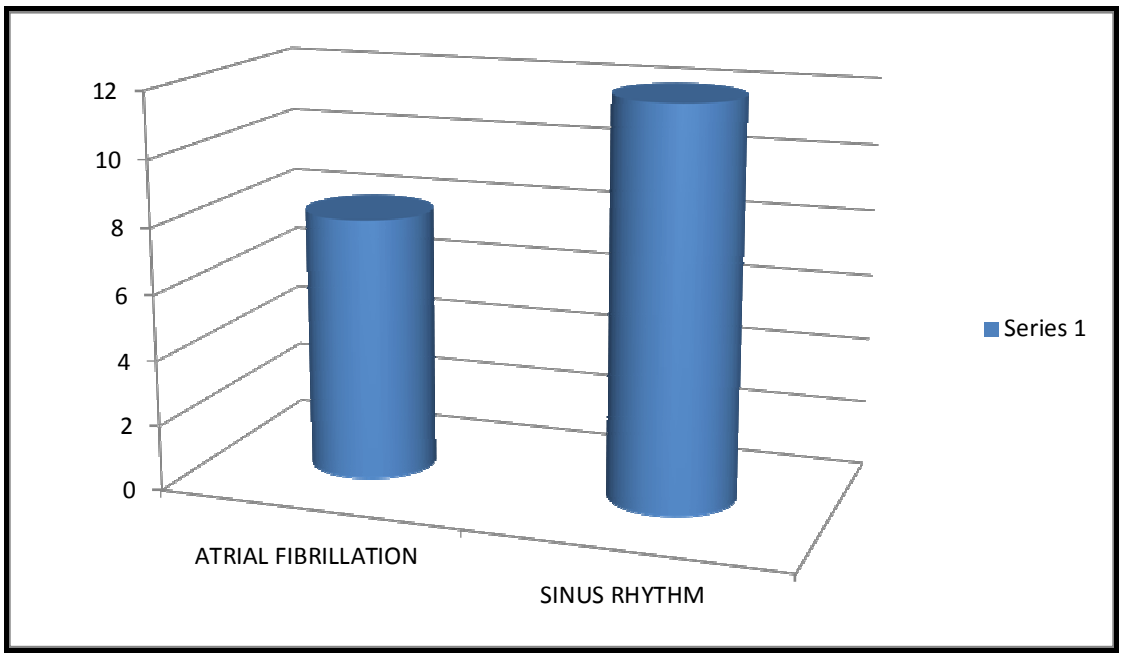


Predominant age of presentation occurs between 25 to 30 yrs as evidenced by the bar chart[histogram- figure -6.]

ATRIAL FIBRILLATION IN THE STUDY GROUP

Out of 20 patients presented with mitral stenosis, 12 patients were in sinus rhythm and 8 patients were in atrial fibrillation under controlled ventricular rate. This is well depicted in bar diagram in figure-3. This necessitated to take several measurements before reporting the values.

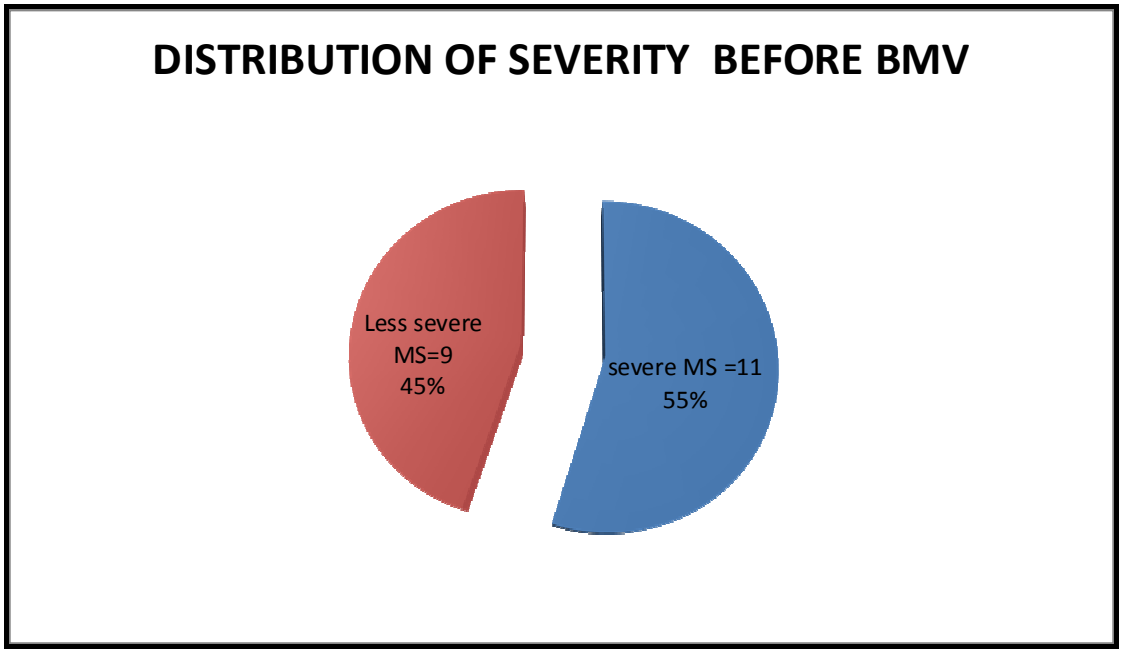
Figure-8: Bar diagram representing prevalence of atrial fibrillation in PTMC candidates.



BASELINE ECHO PARAMETERS

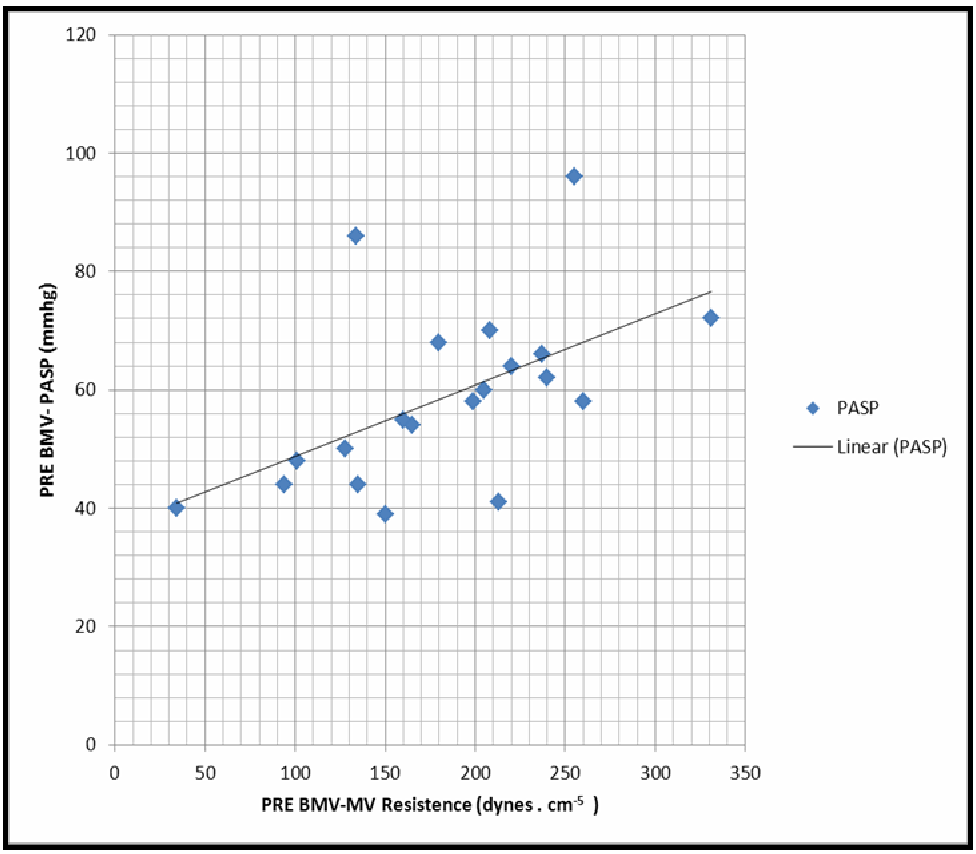
The mean left atrial diameter measured in parasternal long axis in patients before and after percutaneous mitral valvotomy were 4.8±0.61 and 3.9±0.59 respectively. The mean mitral valve area measured by planimetry before and after valvotomy were 1.02±0.2 and1.5±o.23 respectively. Similarly the mean mitral valve area determined by pressure half time before and after transmitral ballon commissurotomy was 1.06±0.23 and1.65±0.64 respectively. The severity assessed by mean mitral gradient at baseline was 14.9±3.3It was decreased to 7.3±1.7 which denotes 50% drop in gradient across mitral valve.

Figure-9 showing the distribution of severe and less severe MS before BMV.



The stroke volume calculated by continuity equation before and after commissurotomy have shown significant differences such as , the mean value of stroke volume were 52±14 ml/sec and 62±16 ml/sec., nearing 20% increase .

Figure-10: Linear regression analysis showing the correlation between PASP and mitral valve resistance before BMV.



Pulmonary artery systolic pressure estimated by tricuspid regurgitation jet had a mean value of 58.75±15, which subsequently after transmitral commissurotomy decreased to 49±10.3.

The most important parameter as for as this study is concerned, the mitral valve resistance measured before valvotomy as a mean value was 182.4±68, which after transcatheter mitral intervention declined to 74±35.3. All these changes in echocardiographic parameters are shown in table-1 and table-2

CORRELATION BETWEEN PASP AND OTHER ECHO VARIABLES

CORRELATION BETWEEN PASP AND OTHER ECHO VARIABLES BEFORE PTMC: [SPEARMANS ANALSIS]

Table-3: Correlation of systolic PAP with other echo variables. r Pearson coefficient and p denotes significance before PTMC.

VARIABLES	r	p
STROKE VOLUME[ml/s]	-0.273	0.001
LA DIAMETER	-0.008	0.973
MVA[P]	-0.602	0.001
MVA[PHT]	-0.697	0.005
TMMG[mmhg]	0.607	0.013
MVR[dynes.cm-5	0.647	0.001

CORRELATION BETWEEN PASP AND OTHER ECHO PARAMETERS AFTER PTMC:

CORRELATION BETWEEN MITRAL VALVE RESISTANCE AND SYSTOLIC PULMONARY ARTERY PRESSURE BEFORE PTMC.

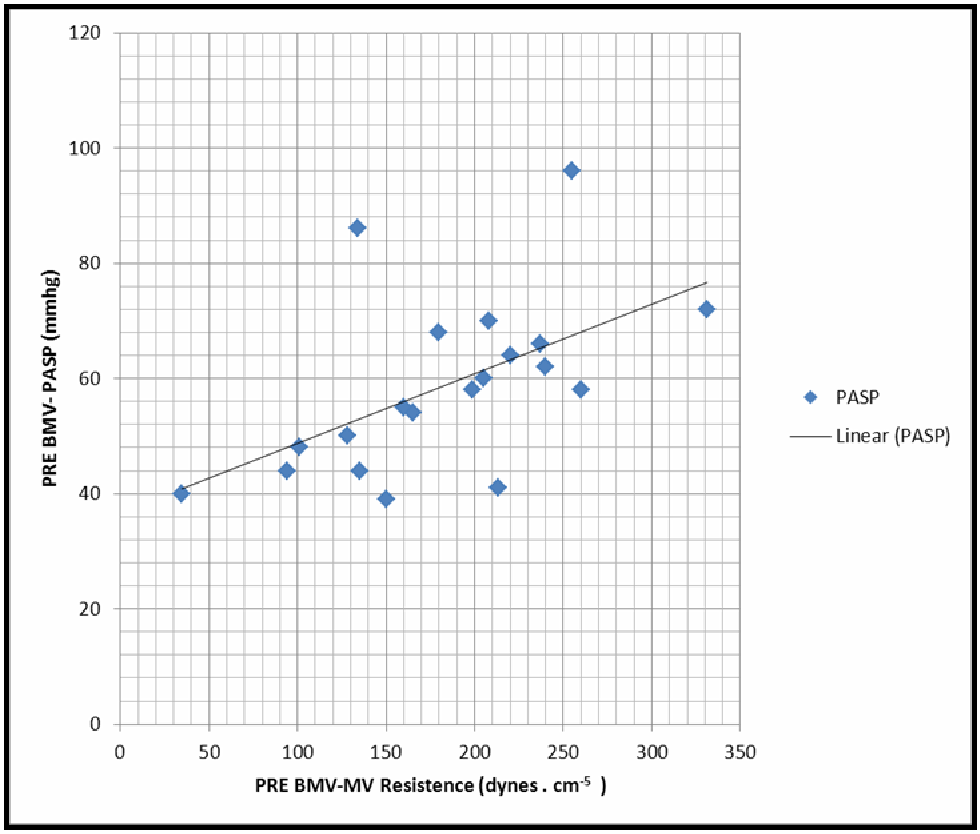
Table-4; Correlation of systolic PAP with other echo variables. r Pearson coefficient and p denotes significance after PTMC.

VARIABLES	r	p
STROKE VOLUME[ml/s]	-0.418	0.140
LA DIAMETER	-0.154	0.518
MVA[P]	-0.613	0.004
MVA[PHT]	-0.519	0.019
TMMG[mmhg]	0.519	0.019
MVR[dynes.cm-5	0.553	0.014

Using spearman's correlation analysis, pulmonary systolic pressure was analysed with mitral valve resistance index, before balloon mitral valvotomy. As shown by the figure 4, there is independent correlation between PASP and MV resistance as evidenced by a highest r value of 0.647, compared with other, and a p value of 0.014. Hence it is independently correlates with mitral valve resistance.

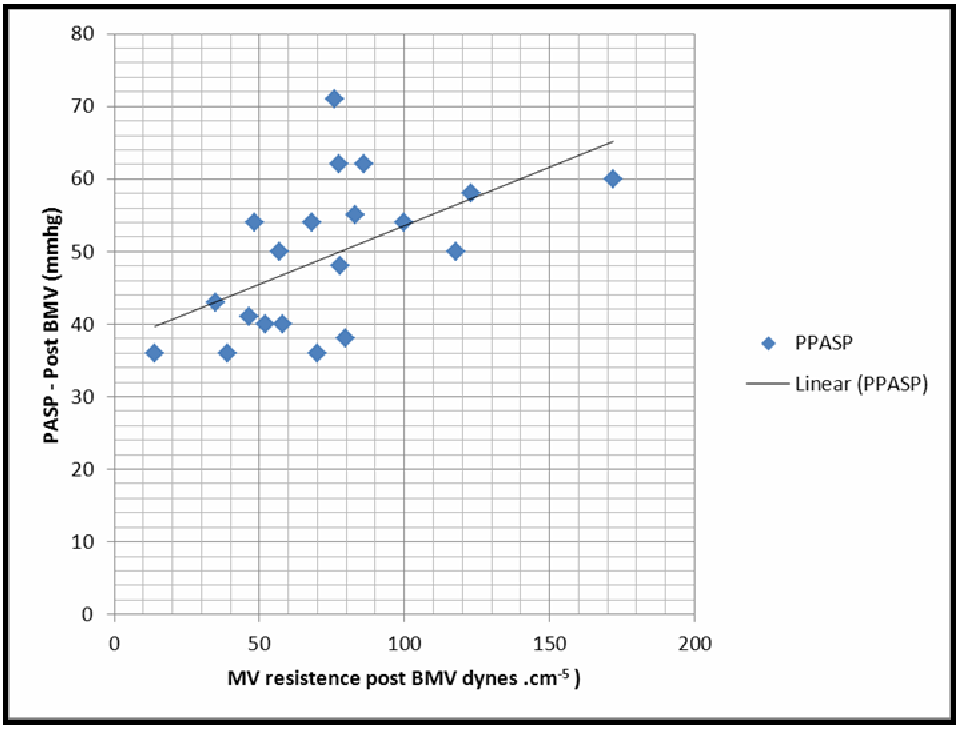
CORRELATION OF PASP AND MV RESITANCE AFTER PTMC:

Figure-11-showing linear regression analysis of correlation between mitral VR and PASP before BMV.



Spearmans spearsons analysis also provides evidence that mitral valve resistance is better correlated with pulmonary artery pressure than other parameters, after BMV as evidenced by highest r value of 0.553, and p value of 0.001. These analysis prove that the correlation is better expressed in patients before PTMC.

FIGURE 12: Linear regression analysis showing the correlation between mitral valve resistance and pulmonary artery systolic pressure after balloon mitral commissurotomy.



MULTIVARIATE ANALYSIS OF MV RESISTANCE AND SYSTOLIC PAP:

Independent association of severity of pulmonary artery systolic pressure is well established by the multivariate analysis done before and balloon mitral valvotomy.

Table-5. Multivariate analysis of valve resistance with PASP as dependent variable, before PTMC.

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	36.758	8.450		4.350	.000
	VR	.121	.043	.547	2.771	.013

a. Dependent Variable: PASP

Table-6;. Multivariate analysis of valve resistance with PASP as dependent variable, after PTMC..

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	37.444	4.685		7.993	.000
	POST BMV VR	.161	.057	.553	2.815	.011

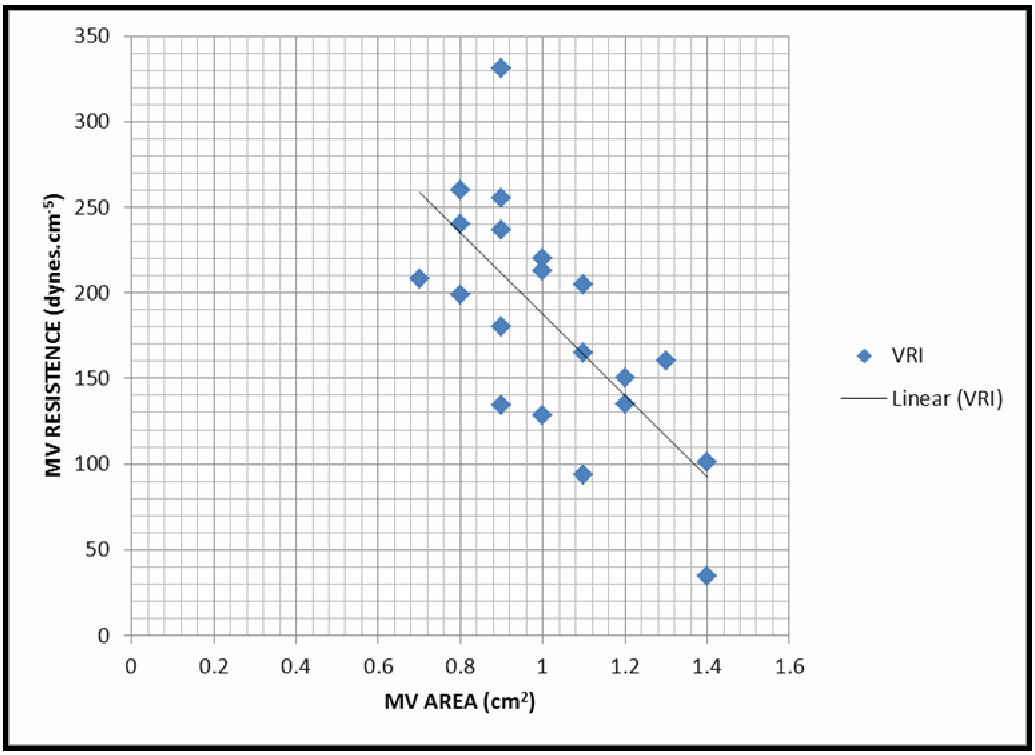
a. Dependent Variable: POST PASP

As shown table 5 and 6, it enables us to understand the mitral valve resistance as the independent determinant of pulmonary artery pressure as the β value before balloon commissurotomy is 0.547 and 0.553 after balloon commissurotomy.

CORRELATION BETWEEN SEVERITY OF MITRAL STENOSIS AND MITRAL VALVE RESISTANCE INDEX

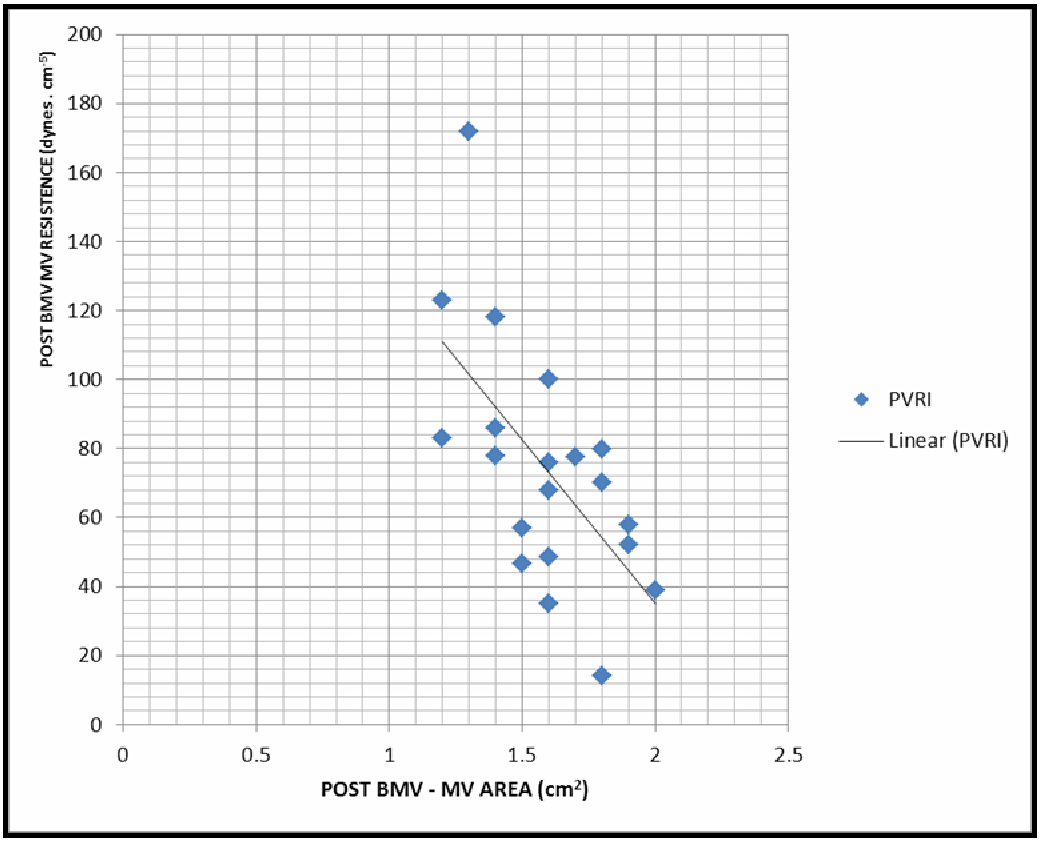
As the valve area decreases, the mitral valve resistance is increasing as evidenced by the figure 7 and figure 8

Figure-13: The linear regression model describing the correlation between the MV area and mitral VR, before BMV.



The change in slope of scatter diagram shows there is inverse relationship between mitral valve orifice area and mitral valve resistance. Both before and after the percutaneous transmitral commissurotomy there is inverse relation between the valve area and resistance index.

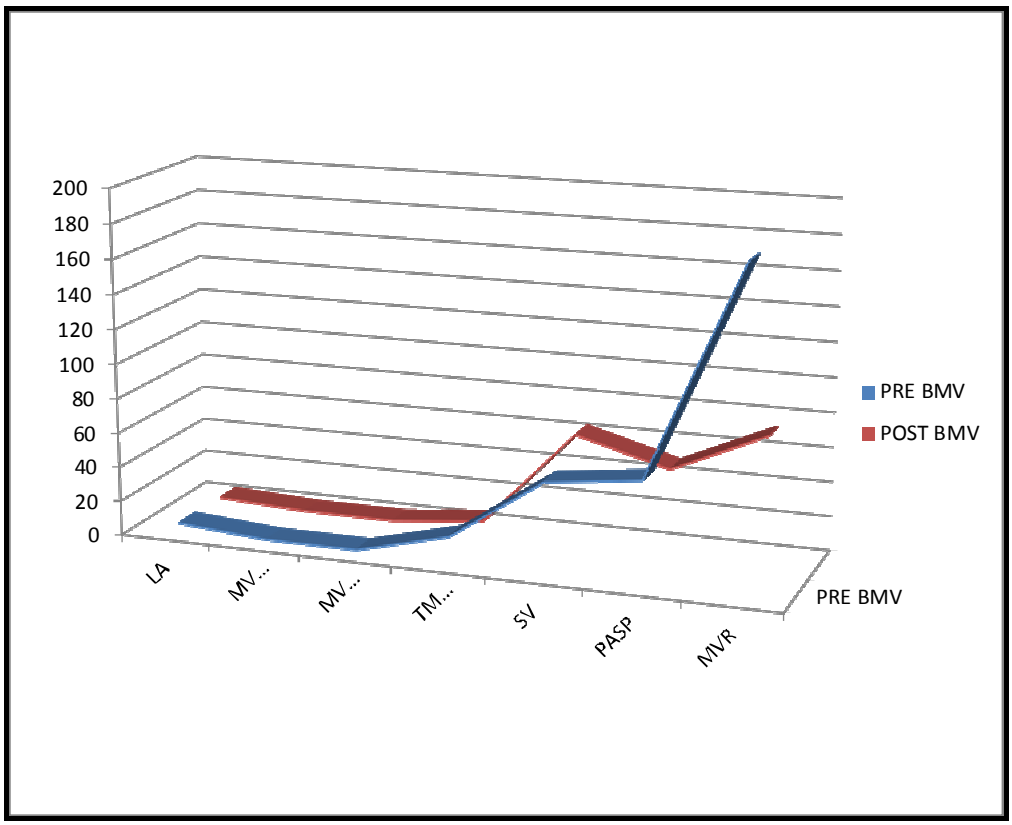
Figure-14; Linear regression model explains the correlation between mitral valve area and mitral VR post BMV.



COMPARISION OF HEMODYNAMICS BEFORE AND AFTER BALLON MITRAL VALVOTOMY

In this study the hemodynamic changes that occur after percutaneous transmitral valvotomy in comparison with pre valvotomy patients are also recorded and analysed. Left atrial diameter decreased from a mean value of 4.8cm^2 to 3.9 cm^2 with a p value of 0.170, denoting less significance. The mean mitral valve area increased from 1.02cm2 to 1.59cm2 with significant p value of 0.001. The mean mitral gradient decreased from 14.9 mmhg to 7.3mmhg amounting to 50% decrease, with significant p value.

Figure-15; Line diagram comparing the echo parameters before and after BMV.



The stroke volume also has significantly increased from a mean value of 52ml/s to 64ml/s as a result of increased filling of left ventricle. Pulmonary artery systolic pressure also significantly dropped from a mean value of 58.7mmhg to 49.4mmhg with a p value of 0.002., consistent with other studies.

Table-7: Echo characteristics of ms before and after PTMC

VARIABLES	Pre PTMC [mean]	Post PTMC [mean]	P value
LA diameter[cm]	4.8	3.9	0.17
MVA[P][cm2]	1.02	1.59	0.001
MVA[PHT][cm2]	1.06	1.65	0.001
TMMG[mmhg]	14.9	7.3	0.000
STROKE VOLUME[ml/s]	52	64	0.001
PASP[mmhg]	58.7	49.4	0.002
MVR[dynes.cm-5]	182	74	0.000

Finally, mitral valve resistance also declined after valvotomy from initial value of 182 dynes. Cm^{-5} to 74 dynes with a p value of 0.000 signifying greater significance

DISCUSSION

This study demonstrates that mitral valve resistance is the most important and the independent predictor of systolic PAP in patients with MS both before and after balloon mitral valvotomy. Elevated PASP is the major hemodynamic consequence of mitral valve obstruction causing the debilitating symptoms, dyspnea, and poor exercise tolerance.1 The major trigger factor for this increased PAP, in mitral stenosis, is the retrograde transmission of increased left atrial pressure to the pulmonary circulation ,resulting in passive pulmonary arterial hypertension, followed by reactive pulmonary hypertension.[19,20]

The results of this study demonstrate that the functional or physiological severity of mitral valve obstruction is better reflected by mitral valve resistance rather than mitral valve area by planimetry or pressure half time method.

MITRAL VALVE RESISTANCE AS A DETERMINANT OF HEMODYNAMIC CONSEQUENCES

Our study clearly demonstrates that the valve resistance is an independent determinant of pulmonary artery pressure as evidenced by the multivariate analysis with the β value of 0.547 and 0.553 and

p value of 0.001 and 0.014 before and after mitral valvotomy respectively. As the hemodynamic consequences of MS are primarily determined by the severity of pulmonary hypertension, valve resistance can be an important adjunct to routine echo evaluation of stenosis.

VALVE RESISTANCE (VR) AS AN INDEX OF MS SEVERITY

It is well known that as valve narrowing worsens, pressure gradient increases but pressure gradient also depends on both amount of blood flow through the valve and the heart rate. Hence ,taking into account both the factors as mentioned above , valve resistance can estimate severity of stenosis with accuracy. It is a expression

of the relation of transvalvular gradient to transvalvular flow across a stenotic valve.[21] VR had been suggested and validated as an index of stenosis long years back,[22,23] but it did not gain much importance. Later studies, however, clearly demonstrated that VR was in fact flow dependent[24]. But, Ford et al,[21] considered VR as a stenotic index due to its higher accuracy in expressing the hemodynamics and impact of stenosis than valve area, despite being flow dependent or independent. The results of this study validate the suggestion of Ford et al[21] in respect to MS.

Particularly, mitral VR was more accurately reflecting the hemodynamic burden of MS than MVA and mean TMG because of its high correlation with resting and stress PAP. Similar results were also observed by Weitzel et al,[25] who also determined mitral VR as the most important independent predictor of resting systolic PAP in a larger group of patients with

MS. They have also established that mitral VR was independently affected by the degree of structural damage of the valve (assessed by Wilkins scoring), which is potentially a contributor to the obstructive effect of the stenotic valve other than its area . Is is also easy to perform this method , and no need to index with body surface area because it takes into account the flow rate also.

VR AS A COMLIMENTARY TOOL TO MVA AND TMMG

Mitral valve area by planimery and pressure half method and mean TMG are commonlyemployed for assessment of severity of mitral stenosis.[26]. Different methods used for MVA calculation have their own well-established intrinsic and technical disadvantages..6,7,9,13[In this regard, the strong correlation between PAP and mitral VR demonstrated in our study suggests

that mitral VR may be useful as a complementary tool to stenotic index in MS [27,28,29].

Mean gradient is considered as a good tool for estimation of MS but hampered by flow dependency and diastolic filling period .In case of severe pulmonary hypertension with severe narrowing , mean gradient may not increase due to reduction in flow through the valve.

USEFULNESS OF VR AFTER BALLOON MITRAL VALVOTOMY

In our study it is clearly evident, that there is strong correlation between VR and systolic PAP 72 hrs after balloon mitral valvotomy as indicated by pearson r was 0.547 with significant p value. Also correlation between mitral vave stenotic severity and VR also established in our study. Hence our study suggests that VR can express physiological severity as well as anatomical severity after balloon mitral valvotomy.

TMMG(TRANSMITRAL MEAN GRADIENT) AS A PREDICTOR OF HEMODYNAMIC STATUS

In our study, after VR , TMMG correlates better with PASP both before and after balloon valvotomy. This is also observed in another study done by Sagie et al, [30] where they found no correlation with severity of stenosis but good correlation with right ventricular hypertension.

LIMITATIONA OF THE STUDY

1) Small sample size

2) Usefulness of valve resistance in therapeutic decision making was not addressed in this study

3) Valve resistance is also flow dependent as suggested by some studies

4) Atrio ventricular compliance was not assessed in this study as it may also affect resting systolic PAP

CONCLUSIONS

1) Mitral valve resistance index is a strong and independent predictor of systolic pulmonary artery pressure both before and after percutaneous balloon mitral valvotomy in mitral stenosis patients.

2) Mitral valve resistance also correlates with severity of mitral stenosis in our study.

3) Because of inherent limitations of conventional echocardiographic parameters in evaluation of MS, valve resistance can be an adjunct tool in assessment of severity.

4) In our study, among conventional indices ,trans mitral mean gradient better correlates with hemodynamic status than mitral valve area.

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ABBREVIATIONS

VR	:	Valvular resistance
PASP	:	Pulmonary artery systolic pressure
TMMG	:	Transmitral mean gradient
PTMC	:	Percutaneous trans mitral commissurotomy
BMV	:	Balloon mitral valvotomy
MVA	:	Mitral valve area
PHT	:	Pressure half time
DFP	:	Diastolic filling period
LA	:	Left atrium
SV	:	Stroke volume
MS	:	Mitral stenosis

Figure-15 ECHO recording showing pressure half time calculation in mitral stenosis.

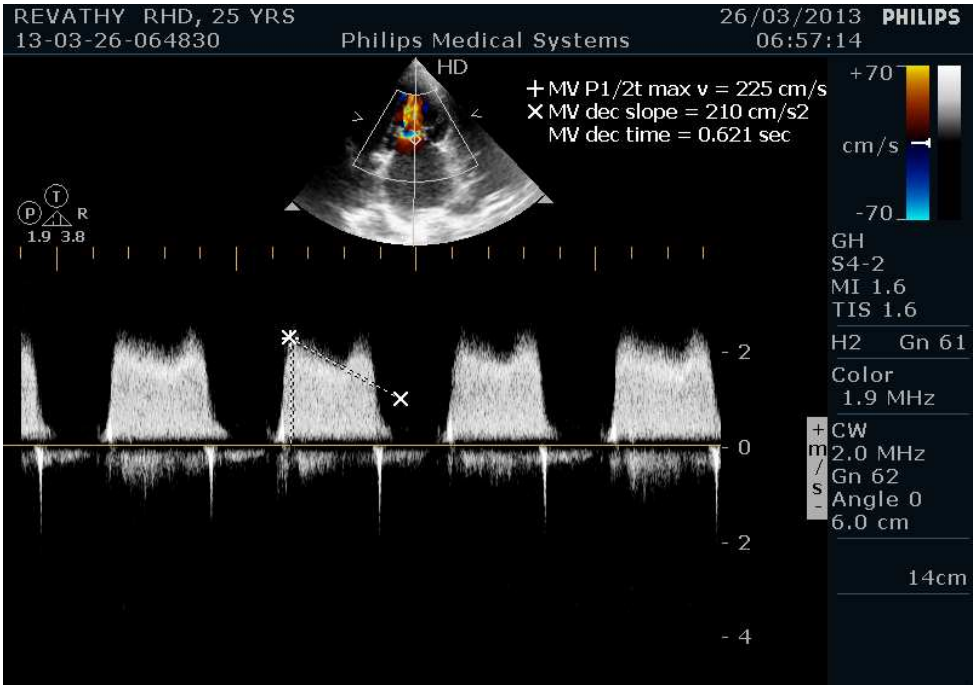


Figure-16; Echo recording showing transmitral pressure gradient estimation.

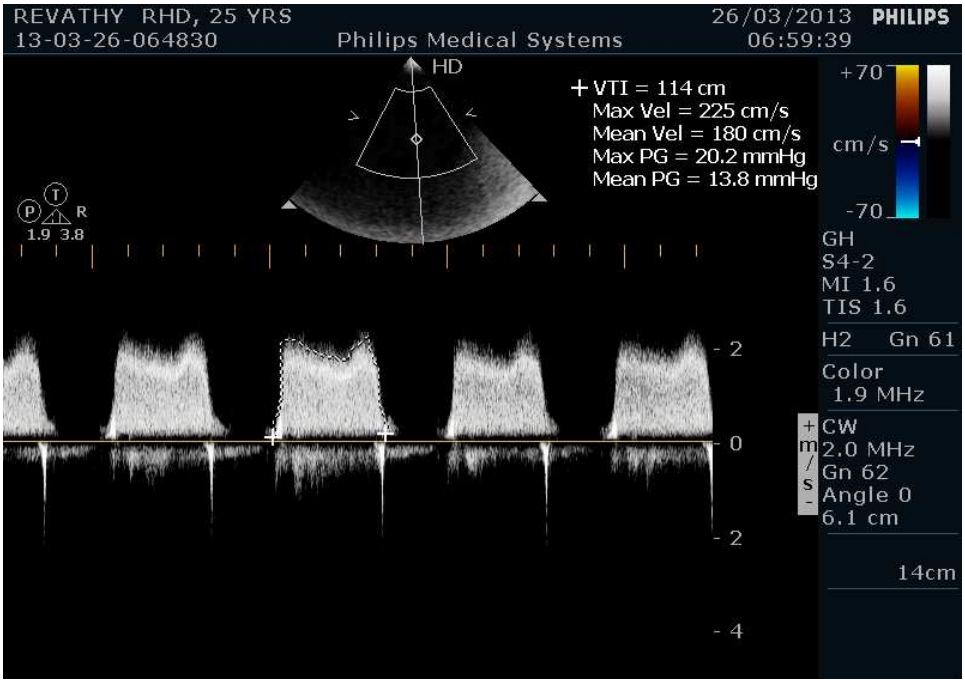


Figure-17: Echocardiographic recording of planimetry of mitral valve area in mitral stenosis.

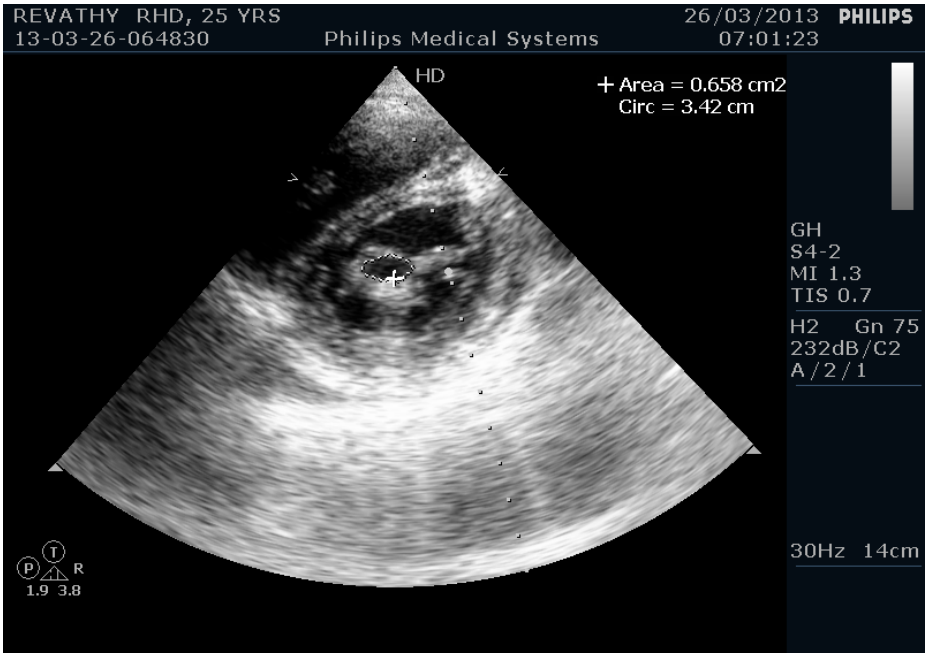
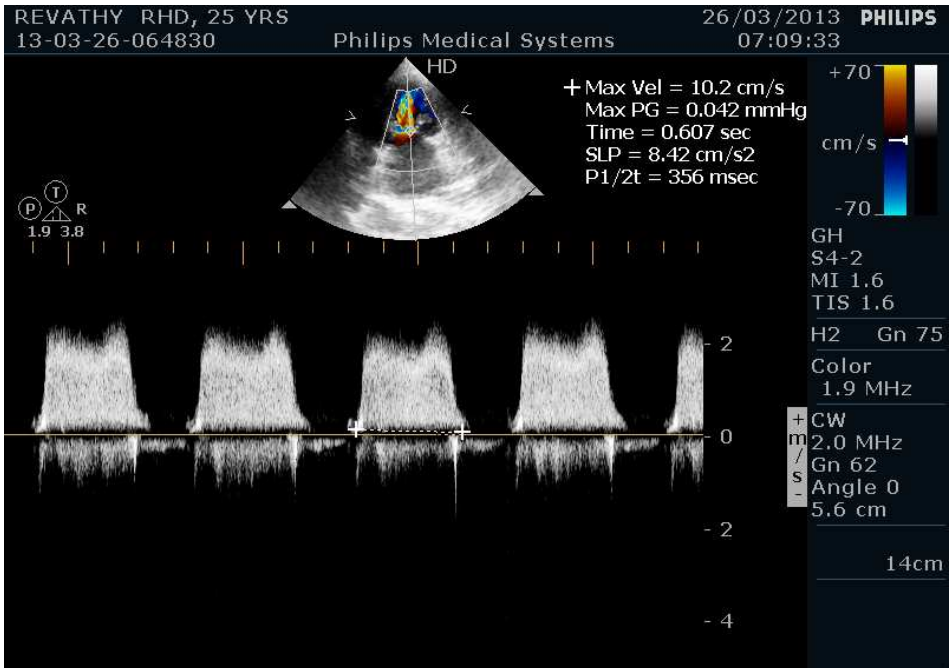


Figure-18; diastolic filling period in mitral stenosis by echocardiography.



ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு: மைட்ரல் வால்வு சுருக்க தன்மையை பலூன் விரிவாக்கச் சிகிச்சைக்கு முன்பும் பின்பும் மின் ஒலி இதய வரைவு மூலம் ஆய்வு செய்தல்.

பெயர் : **தேதி :**

வயது : **நோயாளி எண் :**

பால் : **ஆராய்ச்சி சேர்க்கை எண் :**

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன்.

எனக்கு மின் ஒலி இதய வரைவு (மார்பகதாண்டு) பரிசோதனை செய்து கொள்ள சம்மதம்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும், அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் புரிந்து கொண்டேன்.

நான் மைட்ரல் வால்வு சுருக்க தன்மையை பலூன் விரிவாக்கச் சிகிச்சைக்கு முன்பும் பின்பும் மின் ஒலி இதய வரைவு மூலம் ஆய்வு மேற்கொள்ளப்படும் இந்த ஆராய்ச்சியின் விபரங்களைக் கொண்ட தகவல் தாளைப் பெற்றுக் கொண்டேன்.

இதன் மூலம் எந்த பின்விளைவும் வராது என மருத்துவர் மூலம் தெரிந்து கொண்டு என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

கையொப்பம்

ஆராய்ச்சி தாள்

சென்னை அரசு பொது மருத்துவமனைக்கு வரும் நோயாளிகளிடம் மைட்ரல் வால்வு சுருக்க தன்மையை பலூன் விரிவாக்கச் சிகிச்சைக்கு முன்பும் பின்பும் மின் ஒலி இதய வரைவு மூலம் ஆய்வு செய்தல். என்ற ஆராய்ச்சி இங்கு நடைபெற்று வருகின்றது.

பலூன் விரிவாக்கச் சிகிச்சை மூலம் மைட்ரல் வால்வு விரிவாக்கம் பெற்ற நோயாளிகளுக்கு ஒலி இதய வரைவி மூலம் இதய செயல்பாட்டை ஆய்வு செய்வதன் மூலம் இதய பாதிப்பு தீவிரத்தன்மை பற்றி அறிய முடியும் .மின் ஒலி இதய வரைவு (மார்பகதாண்டு) மூலம் இதய மைட்ரல் வால்வு சுருக்க தன்மை எவ்வாறு சீராகியுள்ளது என்பதை மதிப்பாய்வு செய்தலே இவ்வாரய்ச்சியின் நோக்கமாகும்.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம் . அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்புக்கு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறோம்

. இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது.மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துகொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

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Author	Rajendran Manickam 16101509 D.M. Cardiology
E-mail	docsamniha@yahoo.co.in
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INTRODUCTION Valvular stenosis is common cardiac disease with greater morbidity and mortality especially in developing countries like India. Echocardiography is considered as an important and simple tool to evaluate valve stenosis .Almost cases of mitral stenosis are due to rheumatic heart disease. Assessment of severity of mitral stenosis by echocardiography utilizes many parameters using 2D echo, M mode, and Doppler methods. Conventional methods include mitral valve orifice area determination by planimetry and pressure half time method, pressure gradient determinations with Bernoulli's equation, and mitral leaflet separation index. But the common problem that occurs in all these...

INTRODUCTION

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PRE BMV

S.No	AGE	SEX	LA D]	MVA	PHT	MG	LVOT VTI	LVOT D	STROKE V	DFP	PASP	VRI
1	30	M	4.2	0.9	0.8	21	17.7	16	35.5	420	72	331
2	35	M	4.4	1.2	1.5	14	18	19	51	412	39	150
3	29	F	4.7	0.8	0.8	19.2	17.9	19	49	382	58	199
4	25	F	5.2	0.7	0.9	17	18.4	19	52	479	70	208
5	42	F	5.2	1	1.2	16	25	16	50	502	41	213
6	39	F	4.9	1.1	1.3	11	19	17	43	486	54	165
7	27	M	5.1	1.2	1.2	12	23	16	46	390	44	135
8	27	F	5.5	0.9	1.1	15	17.8	17	40	478	66	237
9	36	F	5.4	1	1.2	16	19	18	48	500	64	220
10	42	M	50	0.8	1.2	18	21	16	42	458	58	260
11	45	F	4.7	1.4	1.4	8.9	28.2	21	96	282	40	34.5
12	33	F	3.9	1.1	1.1	12.4	19	16	38	475	60	205
13	29	M	4.4	0.9	0.9	18	27.8	18	70	398	86	134
14	36	F	5.2	1.1	1.2	11	19	21	65	420	44	94
15	30	F	4.9	1.4	1.1	13	23	19	65	380	48	101
16	28	F	5.3	1.3	0.9	16	25	18	63	396	55	160
17	26	M	4.8	1	1.3	11	18	20	56	488	50	128
18	29	M	5.5	0.9	0.8	19	22	17	49	500	96	255
19	27	M	5	0.9	0.7	18	20	17	45	398	68	180
20	37	F	5.1	0.8	0.7	13	20	16	40	556	62	240

POST BMV

1	3.8	1.3	0.6	11	19	16.8	41	483	60	172
2	3.6	1.5	1.5	9	20	21	69	267	41	46.5
3	4	1.6	1.1	7	28	16	56	408	54	68
4	2.5	1.2	1.4	8.4	20	20	62	460	55	83
5	4.4	1.8	1.7	7	26	17	58	500	38	79.7
6	3.9	1.4	1.1	8	21	17.7	51	370	48	77.8
7	4.6	1.8	2	5.6	25	16	55	521	36	70
8	4.8	1.6	1.4	6	25	17	59	360	54	48.5
9	4.8	1.5	1.8	6	21	19	59	421	50	57
10	4.3	1.4	1.7	9	21	17	50	490	50	118
11	3.9	1.8	2	3.2	29	22	111	370	36	14
12	2.8	1.6	1.6	7.5	21	17	47	470	54	100
13	3.6	1.7	2.1	9	29	18	81	520	62	77.4
14	4.2	1.9	2.3	6	21	22	79.7	580	40	58
15	3.7	1.9	1.9	8	25	20	78.5	380	40	52
16	4.7	1.6	1.9	7	28	19	79	298	43	35
17	3.6	2	2.5	6	19	22	72	360	36	39
18	4.1	1.6	1.7	7.8	25	18	63	466	71	76
19	4.2	1.4	1.3	9	24	17.8	59.6	427	62	86
20	4	1.2	1.4	6.2	23	17	54	500	58	123

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.M.Rajendran,
DM Cardiology,
RGGGH & MMC,
Chennai -3.

Dear Dr.M.RAJENDRAN

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Assessment of mitral valve Resistance index by Echocardiography in mitral stenosis before and after Baloon mitral valvotomy and its Hemodynamic implications" No.06032013.

The following members of Ethics Committee were present in the meeting held on 05.03.2013 conducted at Madras Medical College, Chennai -3.

1. Dr.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. A. Radhakrishnan MD -- Member
   Prof of Internal Medicine, MMC, Ch-3
6. Prof. S. Deivanayagam MS -- Member
   Prof of Surgery, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandhini

Member Secretary, Ethics Committee

PROFORMA

S.NO		MRD NO:
1	Patient Name	
2	Age (in years)	
3	Sex	1. Male 2. Female
4	Education	
5	Name of Hospital	
6	Contact number	
7	Address:	
8	Name of respondent (patient or attendant)	
9	Duration of symptoms	
10	Date of admission	
11	Date of Discharge	
12	Clinical examination	Weight Height BMI Body surface area
13	Electrocardiogram	HR/mt Sinus rhythm
	ECHO CARDIOGRAM	BEFORE PTMC
14	LVD- ESD/EDD/EF	

15	Mitral valve area [MVA]cm2 by planimetry	
16	Mitral valve area by pressure half time [MVA]cm2	
17	Mean pressure gradient [mmhg]	
18	Mean pulmonary artery systolic pressure [mmhg]	
19	Stroke volume = LVOT TVI×LVOT CS Diastolic filling period [msec]	